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Meconium peritonitis following intestinal atresia: A case report

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ABSTRACT

Meconium peritonitis is a sterile chemical peritonitis, which frequently occurs after intestinal perforation *in utero*. Overall mortality rates have drastically decreased with earlier prenatal diagnosis and improved perinatal care. However, perinatal surgical management of meconium peritonitis is largely dependent on individual surgeon experience. We present a case of meconium peritonitis with emergent cesarean section delivery after the patient developed massive meconium ascites, hydrops fetalis, and non-reassuring fetal monitoring. In the immediate post-natal period, the patient was intubated and a peritoneal drain was placed for respiratory and hemodynamic stabilization. He was then taken to the operating room the following day for laparotomy and bowel resection. His post-operative course was uneventful, and he was discharged home in good clinical condition.

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Meconium peritonitis (MP) is a sterile chemical peritonitis caused by intestinal perforation *in utero* and carries an incidence rate of 1 in 30–35,000 births [1]. Historically, mortality rates of 60–90% have been reported; however, advances in perinatal care and surgical management have improved survival rates to 80–92% [1–3].

Despite the increasing numbers of prenatally diagnosed cases of MP, surgical management is largely dependent on fetal imaging. We present a case of MP, which presented as massive meconium ascites delivered emergently due to acute fetal decompensation, followed by bedside stabilization and peritoneal drainage. Surgical exploration was performed the following day with good clinical outcomes.

1. Case report

A 41-year-old G2P1 female, in her 28th gestational week of pregnancy complicated by gestational diabetes and Trisomy 21 (diagnosed at 13 weeks by chorionic villus sampling and amniocentesis; karyotype 46, XY/47, XR, +21), was discovered to have new fetal ascites and dilated bowel on routine sonography at 28 weeks. She had previously underwent sonogram at 20 weeks, which was normal. At 32 weeks, sonography demonstrated markedly increased ascites and now collapsed bowel, suspicious for intestinal perforation. Expectant management continued and imaging at 33 weeks and 5 days revealed worsening polyhydramnios, massive ascites, hyperextension of the fetal neck, new fetal pericardial effusion—concerning for hydrops fetalis. Fetal monitoring was non-reassuring and emergent cesarean section was elected. Apgar scores were 6 at 1 min and 8 at 5 min of life, and the patient was immediately intubated for respiratory distress. The abdomen was distended and tense and likely contributing to respiratory distress, thus an 8.5 French pigtail abdominal drain was placed immediately at the bedside following birth with removal of 100 mL of bilious ascites. The patient remained in the neonatal intensive care unit overnight, with gradual removal of an additional 240 cc of ascites over the following 12 h and weaning of respiratory support.

The following day, the patient was taken to the operating room for exploration. Approximately 200 mL of bilious ascites was evacuated upon entering the abdomen. The large bowel was identified and appeared normal and collapsed. The small bowel was run proximally until the mid-jejunum, where a severely narrowed segment with proximal dilation and perforation was encountered, consistent with intestinal atresia. Approximately 15 cm of non-viable bowel was resected (Fig. 1) and an anastomosis with a distal Cheatle slit to minimize size discrepancy was performed.

Post-operatively, the patient remained on dopamine for hemodynamic support, which was weaned off by post-operative day 3. His respiratory status continued to improve and he was extubated on post-operative day 5. He regained bowel function on post-operative day 7 and was started on low volume enteral feeds with gradual advancement. The remainder of his hospital course was

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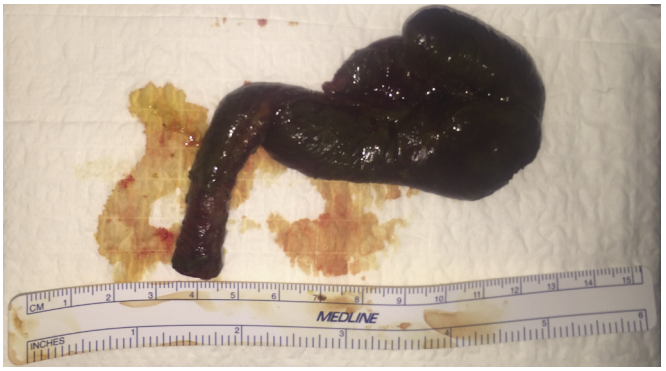

Fig. 1. Excised segment of non-viable bowel including atretic segment and overlying meconium ascitic fluid.

uneventful and his discharge weight was 2475 g (birth weight 3010 g) at 28 days of life. He has since been followed up at 42 days of life and was doing well with normal weight gain, tolerating feedings, and meeting developmental milestones.

2. Discussion

MP occurs in 1 in 30,000–35,000 births and is increasingly diagnosed *in utero* [1]. The majority of cases reported are due to intestinal atresia, though other etiologies have been described [1,3–9]. Historical mortality rates are 60–90%, however with earlier diagnosis and improvements in perinatal care, survival rates have increased to 80–92% [2,3,10,11]. Individual outcomes are influenced by the severity of MP seen on prenatal ultrasound, gestational age, clinical status of the patient after delivery, and timing of surgical management [5,12].

Prenatal ultrasound demonstrating fetal ascites, intra-abdominal calcifications, and dilated loops of bowel are most frequently associated with MP [3]. Additionally, the presence of intestinal pseudocysts and isolated polyhydramnios are almost pathognomonic [3,6,10,12]. In terms of the need for surgical management, Shyu et al. [10] found persistent ascites, pseudocyst or dilated bowel to be associated with the need for post-natal surgery. Other authors believe pseudocysts and massive meconium ascites indicate severe intestinal obstruction and require early and emergent surgery [6,8]. In our case, the patient demonstrated persistent fetal ascites and bowel dilation with subsequent collapse between her 28th and 32nd gestational weeks. He remained clinically stable during this period and therefore prenatal surveillance continued without induction of preterm delivery or surgical intervention until fetal distress became obvious.

It is important to note that if the fetus remains stable, induction should be avoided as MP may resolve *in utero*, and increased gestational age is associated with better outcomes [3]. Certainly, persistent massive ascites causing pulmonary hypertension and hypoplasia requires decompression and may necessitate earlier delivery or fetal instrumentation. Okawa et al. [13] reported a case of massive meconium ascites managed with repeated fetal paracentesis from 28 to 36 weeks gestation.

After delivery, surgical intervention within the first 24 h of life is advisable. After 72 h, 86% of patients will have intestinal bacterial colonization and develop overwhelming sepsis with mortality rates of 75%, while only 24% of meconium cultures are positive within the first 12 h of life with associated mortality rates of 10% [14].

In our patient, an abdominal drain was placed within the first minutes of life and bilious ascites was removed at an hourly rate with replacement fluids in order to decrease the effect of fluid shifts between compartments. Fluid was gradually removed to decrease hemodynamic collapse and physiologic strain. The following day, he was brought to the operating room for exploration, bowel resection, and primary anastomosis, and had a favorable outcome following this approach.

3. Conclusion

Meconium peritonitis is increasingly diagnosed earlier in gestation. Timing of delivery is based on whether the case is simple or complex, and then observing the development of hydrops fetalis or other grave signs of fetal danger. In our experience, newborns with significant ascites can be treated with bedside abdominal drainage for stabilization, relief of respiratory distress and hemodynamic instability, and preparation for operation.

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